



Using science to create a better place

Soil Guideline Values for inorganic arsenic in soil

Science Report SC050021/ arsenic SGV

This technical note is one in a series that describe Soil Guideline Values (SGVs) for individual, or groups of similar, chemicals to assist in the assessment of risks from land contamination.

SGVs are an example of generic assessment criteria (Defra and Environment Agency, 2004) and can be used in the preliminary evaluation of the risk to human health from long-term exposure to chemicals in soil. Specifically, this note provides SGVs for inorganic forms of arsenic in soil. It does not include organic arsenicals, which are considered to be less toxic to humans than inorganic forms.

The SGVs and the additional advice found here should be used only in conjunction with the introductory guide to the series entitled *Using Soil Guideline Values* (Environment Agency, 2009a), the framework documents *Updated technical background to the CLEA model* (Environment Agency, 2009b) and *Human health toxicological assessment of contaminants in soil: updated collation of toxicological data and intake values for humans. Inorganic arsenic* (Environment Agency, 2009d). Supplementary information on arsenic is also available (Environment Agency, 2009e).

All notes in the SGV series, the introductory guide and further supplementary information can be downloaded from our website (<http://www.environment-agency.gov.uk/clea>).

Arsenic and its compounds

In its elemental form arsenic (CAS No. 7440-38-2) occurs in two forms under ambient conditions – a steel grey coloured brittle metallic solid or a dark grey amorphous solid (ATSDR, 2007). Although it is commonly described as a heavy metal, arsenic is a metalloid with a complex chemistry similar to phosphorous (CCME, 2001).

Arsenic occurs naturally in the environment although rarely in its elemental form (CCME, 2001). Over 200 arsenic-containing minerals have been identified, with approximately 60 per cent being arsenates, 20 per cent sulphides and sulphosalts, and the remaining 20 per cent including arsenides, arsenites and oxides (O'Neill, 1995). The most commonly occurring form is arsenopyrite, an iron arsenic sulphide associated with many types of mineral deposits and especially those including sulphide mineralisation (O'Neill, 1995; Farago *et al.*, 2003).

Arsenic forms organic and inorganic compounds with the most common valence states being -3, +3 or +5 (ATSDR, 2007). Arsenic trioxide (CAS No. 1327-53-3) is a white crystalline solid at room temperature. It is produced commercially as a by-product of the smelting of non-ferrous ores including copper and lead (IPCS, 2001; ATSDR, 2007). Most arsenic trioxide is subsequently converted to arsenic acid (H_3AsO_4), which forms arsenate salts (ATSDR, 2007).

Due to its known toxicity, use of arsenic in many applications has either been banned or phased out (ATSDR, 2007). Most arsenic is currently used to produce copper chrome arsenate (CCA), a wood preservative and pesticide (ATSDR, 2007). In 2003, the UK was reported to be the largest consumer of CCA in the European Union at 15,000 tonnes per year and the location of three of the four manufacturing plants (Anon., 2003). As a result of an EU directive, use of CCA is now restricted in the UK to specified formulations and timber uses.¹ High purity arsenic is also used in the manufacture of gallium arsenide semi-conductors, which are used in telecommunication systems, solar cells and space research (ATSDR, 2007).

Historically, inorganic arsenic compounds including calcium arsenate, lead arsenate and sodium arsenite have been used as pesticides. In particular, lead

¹ The Controls on Dangerous Substances and Preparations (Amendment) Regulations 2007 (SI 2007 No. 1596)
http://www.opsi.gov.uk/si/si2007/uksi_20071596_en_1

arsenate has been used for pest control in fruit orchards (ATSDR, 2007).

Arsenic and its inorganic compounds have also been used as a decolouriser in the manufacture of glass, in various metallurgical processes including the production of alloys, in veterinary and human medicines, and lead-acid batteries (CCME, 2001; IPCS, 2001; ATSDR, 2007; Kabata-Pendias and Mukherjee, 2007).

Potential harm to human health

The principles behind the selection of Health Criteria Values (HCVs), and the definition of concepts and terms used, are outlined in *Human health toxicological assessment of contaminants in soil* (Environment Agency, 2009c). Specific information on the toxicity of arsenic and its compounds has been reviewed in *Contaminants in soil: updated collation of toxicological data and intake values for humans. Inorganic arsenic* (Environment Agency, 2009d) and only a brief summary is presented here.

Although arsenic can exist in different oxidation states and has a number of different inorganic and organic compounds, most toxicity data relate to arsenic in its inorganic form. Therefore, inorganic arsenic alone has been considered in the selection of HCVs (Environment Agency, 2009d). Despite an extremely limited database, organic arsenic compounds are generally considered to be less toxic.

Inorganic arsenic is carcinogenic in humans (Environment Agency, 2009d). Long-term exposure produces lung tumours via inhalation and a range of cancers via the oral route (most clearly cancer of the skin, bladder and lung). Absorption through the inhalation route depends primarily on the particle size and solubility.

Table 1

Recommended Health Criteria Values for inorganic arsenic (Environment Agency, 2009d)¹

Parameter	Arsenic
ID _{oral} , $\mu\text{g kg}^{-1} \text{ bw day}^{-1}$	0.3 ²
ID _{inh} for deriving SGV, $\mu\text{g kg}^{-1} \text{ bw day}^{-1}$	0.002

Notes ¹ MDI data are not reproduced here as the HCVs for arsenic are Index Doses.

² Based on the UK drinking-water standard for arsenic.

bw = bodyweight

ID = Index Dose

HCVs for inorganic arsenic are summarised in Table 1. The oral Index Dose (ID_{oral}) in Table 1 is based on the UK drinking-water standard for arsenic. This dose is estimated to be associated with an excess lifetime

systemic cancer risk of around 40 to 400 in 100,000 (Environment Agency, 2009d).

An ID_{oral} derived on the basis of minimal risk in accordance with the principles described in the toxicological framework report (Environment Agency, 2009c), would lie in the range of 0.0006 to 0.003 $\mu\text{g kg}^{-1} \text{ bw day}^{-1}$. However, the UK drinking water standard for arsenic of 10 $\mu\text{g L}^{-1}$ is equivalent to a higher intake of approximately 0.3 $\mu\text{g kg}^{-1} \text{ bw day}^{-1}$ (Environment Agency, 2009d).

In accordance with *Guidance on the legal definition of contaminated land* (Defra, 2008), the toxicological framework report (Environment Agency, 2009c) notes that:

“If a guideline for a non-threshold carcinogen has been produced under a different regulatory regime with UK jurisdiction that is less stringent than the derived ID [Index Dose], it may be considered disproportionate to enforce a stricter limit for contaminated land, and therefore inappropriate to set the SGV [Soil Guideline Value] on the derived ID. In such instances, the ID and SGV may be set based on equivalence to the existing guideline.”

This is the case for the ID_{oral} for arsenic used in the derivation of the SGVs in this report.

The inhalation Index Dose (ID_{inh}) in Table 1, which is based on the World Health Organization (WHO) air quality guidelines for Europe, is estimated to be associated with an excess lifetime lung cancer risk of 1 in 100,000.

For adults, background inhalation exposure to inorganic arsenic from its presence in ambient air is estimated to be approximately 0.014 $\mu\text{g day}^{-1}$. The background oral exposure from its presence in food and drinking-water is much higher at 5 $\mu\text{g day}^{-1}$ (Environment Agency, 2009d).

Exposure assessment

Occurrence in soil

Arsenic occurs naturally in soils as a result of the weathering of the parent rock (O'Neill, 1995). Although it occurs in igneous rocks, the greatest concentrations tend to be found in argillaceous sedimentary rocks (e.g. shales and mudstones) and in heavily sulphidic mineralised areas (O'Neill, 1995; Kabata-Pendias and Mukherjee, 2007).

Anthropogenic activity has resulted in the widespread atmospheric deposition of arsenic from the burning of coal and the smelting of non-ferrous metals including copper (O'Neil, 1995). Agricultural practice including the historical use of arsenic-based pesticides and ongoing application of fertilisers, sludges and manures containing arsenic has resulted in the accumulation of arsenic in topsoils (O'Neil, 1995; Kabata-Pendias and Mukherjee, 2007). In parts of Devon and Cornwall, a long history of mining tin, copper and arsenic has resulted in soil and

water pollution arising from mine tailings and other wastes (Xu and Thornton, 1985; Thornton, 1994).

The Soil and Herbage Survey (UK SHS) is a comprehensive survey of the concentrations of major contaminants in soils and herbage across the UK. The UK SHS found total arsenic concentrations in the range of 0.5 to 143 mg kg⁻¹ dry weight (DW) for rural soils, with a mean of 10.9 mg kg⁻¹ DW (Environment Agency, 2007a). There was considerable geographical variation across the UK, with higher mean concentrations in England and Wales (13.9 and 14.7 mg kg⁻¹ DW respectively) than in Scotland and Northern Ireland. Although the UK SHS reported a mean arsenic concentration for urban soils (11 mg kg⁻¹) similar to rural levels, the reported range from 1.75 to 32 mg kg⁻¹ was much narrower (Environment Agency, 2007a).

Natural geology and the impact of extensive mining activity have resulted in elevated arsenic levels in several regions of the UK. Xu and Thornton (1985) reported a median arsenic concentration in garden soils in the town of Camborne, Cornwall, of 320 mg kg⁻¹ DW, the result of extensive regional mining activity. In Northamptonshire and Lincolnshire, elevated soil concentrations of total arsenic are associated with the underlying ironstone lithology (Nathanail *et al.*, 2004, 2006; Palumbo-Roe *et al.*, 2005).

Behaviour in the soil environment

Recommended values for chemical data used in the exposure modelling of arsenic and its inorganic compounds are shown in Table 4. Data for arsine are not provided and exposure to arsine has not been included in the derivation of the SGV in Table 2. Further information about the selection of chemical properties and the derivation of the soil-to-plant concentration factors can be found in *Supplementary information for the derivation of SGVs for arsenic* (Environment Agency, 2009e).

In typical surface soils, the most important inorganic forms of arsenic are arsenite (AsO₃³⁻) and arsenate (AsO₄³⁻), with the latter dominating under aerobic/oxidising conditions (O'Neill, 1995; Kabata-Pendias and Mukherjee, 2007). It is unusual to find arsenic sulphides in soils, even under waterlogged conditions, because any sulphide mineralisation will have been converted to sulphate and leached out during the weathering process (O'Neill, 1995).

The relationship between arsenate and arsenite in soil and water systems is complicated by the presence of clay minerals, iron and aluminium oxides, organic matter, soil redox potential and pH, and microbial action (O'Neil, 1995; Kabata-Pendias and Mukherjee, 2007). These factors influence the relative stability of the two anions in solution and the rate of oxidation between the two forms in soil.

Arsenite is often considered to be more mobile in soils than arsenate and to be the main species at low pH under reducing conditions (Kabata-Pendias and

Mukherjee, 2007). Both arsenate and arsenite can adsorb to hydrated iron and aluminium oxides, clays and organic matter, although phosphate has been shown to interfere with this (O'Neill, 1995; IPCS, 2001). The role of aluminium oxides and hydroxides in arsenic sorption is significant only in acidic soils (IPCS, 2001). ATSDR (2007) cited the results from a study of 20 Dutch soils which found the most influential factor controlling arsenic sorption in these soils to be their iron content.

Arsenate reportedly binds strongly to iron and manganese oxides, and therefore remains in the surface soil layer after deposition (ATSDR, 2007). Arsenic was observed to be still concentrated after 15 years in the top 20–40 cm of orchard soils treated with lead arsenate (Merwin *et al.* 1994). However, several experimental studies have found that arsenate can be released from iron oxides at alkaline pH as a result of desorption processes (IPCS, 2001; ATSDR, 2007). IPCS (2001) also concluded that carbonates play an increasing role in adsorption of arsenates above a soil pH of 9.

Although arsenic is found in most plants, little is known about its biochemical role (Farago *et al.*, 2003; Kabata-Pendias and Mukherjee, 2007). Excessive uptake of arsenic is considered to disrupt enzyme function and impair phosphate flow in the plant system, with the general tolerance level considered to be around 2 mg kg⁻¹ DW plant tissue (Kabata-Pendias and Mukherjee, 2007).

There has been a significant increase in published studies reporting the arsenic content of fruit and vegetables since publication of the previous SGV in 2002 (Environment Agency, 2009e). There have been several surveys of UK gardens and allotments, and arsenic has been found within all the produce categories considered in the CLEA model (Environment Agency, 2009b).

Dermal absorption of arsenic and its inorganic compounds from soil is not expected to be significant. USEPA (2004) recommended a dermal absorption fraction (ABS_d) of 0.03 for inorganic arsenic and this value has been used in the derivation of SGV.

A review of literature found no data that could be used to provide a generalised arsenic soil-to-dust transport factor. In the absence of a contaminant specific soil-to-dust transport factor, the default value of 0.5 g g⁻¹ DW has been used (Environment Agency, 2009b).

Soil Guideline Values

Soil Guideline Values for arsenic are presented according to land use in Table 2. The SGVs apply only to arsenic and its inorganic compounds. Exposure to arsine gas has not been taken into account in their derivation (see text box overleaf). SGVs have not been derived for organic arsenicals, which are less likely to be present at high concentrations in soil and are generally regarded as being less toxic.

For residential and allotment land uses, SGVs are based on estimates representative of exposure of young children because they are generally more likely to have higher exposures to soil contaminants. Further information on the default exposure assumptions used in the derivation of SGV can be found in *Updated technical background to the CLEA model* (Environment Agency, 2009b).

Analytical limits of detection² for total arsenic concentration in soil will depend on the analytical technique used and range from 0.5 to 3.0 mg kg⁻¹ DW with limits of quantification³ ranging from 2.5 to 15 mg kg⁻¹ DW. Limits of detection and quantification can vary due to the sample matrix and the range, sensitivity and set-up of the instrumentation being used. MCERTS⁴ accredited analytical methods for total arsenic in soil are available.

Table 2

The Soil Guideline Values for inorganic arsenic presented in this table should only be used in conjunction with the information contained in this briefing note and with an understanding of the exposure and toxicological assumptions contained in *Updated technical background to the CLEA model* (Environment Agency, 2009b), *Human health toxicological assessment of contaminants in soil* (Environment Agency, 2009c) and *Contaminants in soil: updated collation of toxicological data and intake values for humans. Inorganic arsenic* (Environment Agency, 2009d).

Land use	Soil Guideline Value (mg kg ⁻¹ DW) ^{1,2,3}
	Inorganic arsenic
Residential	32
Allotment	43
Commercial	640

Notes ¹ Based on a sandy loam soil as defined in Environment Agency (2009b) and 6% soil organic matter (SOM).

² Figures are rounded to one or two significant figures.

³ Based only on a comparison of oral and dermal soil exposure with oral Index Dose.

DW = dry weight

Further risk evaluation

Although the evidence suggests that exposure to arsenic and its inorganic compounds poses a carcinogenic hazard to the lung via both the oral and inhalation routes of exposure (Environment Agency, 2009d), the Soil Guideline Values (SGVs) have been derived based on comparison of the oral and dermal exposure routes with the oral Index Dose (ID_{oral}) only. This approach is appropriate because of the different bases of the oral and inhalation IDs, and the very small contribution that

² Amount of a substance that can be detected but not measured quantitatively

³ Amount present of a substance that can be measured quantitatively

⁴ Environment Agency's Monitoring Certification Scheme

inhalation makes to exposure in the standard land-use scenarios.

As described earlier, the oral Index Dose (ID) has been derived from the current UK drinking-water standard for arsenic and does not represent exposure at minimal risk. The excess lifetime systemic cancer risk associated with the oral ID has been estimated to be between 40 and 400 times higher than a minimal risk level of 1 in 100,000 (Environment Agency, 2009d). The inhalation ID is based on exposures posing a minimal risk. In practice, this means that inhalation exposure to inorganic arsenic equal to the inhalation ID poses a level of risk between 40 to 400 times less than oral exposure equal to the oral ID.⁵

Table 3 presents the estimated contribution via each exposure pathway to total human exposure at a soil concentration equal to the SGV for each land use scenario. Inhalation exposure from indoor and outdoor dust is included for illustrative purposes only and has not been included in the derivation of the SGV. The data show that:

- for the residential and commercial land use scenarios, the ingestion of soil and indoor dust pathway contributes the most to total exposure
- for the allotment land-use scenario, the consumption of home grown produce is the pathway that contributes most exposure with ingestion of soil and indoor dust the second highest pathway

Table 3 also shows that the inhalation of dust makes a very minor contribution to total exposure for all land use scenarios at a soil concentration equal to the SGV and therefore would make a negligible additional contribution to the total cancer risk.

Although the likelihood of an exceedance of the oral ID representing a *significant possibility of significant harm* is much greater than would be the case if the oral ID was based on minimal risk (Environment Agency, 2009d), the SGVs in Table 2 are unlikely to represent a *significant possibility of significant harm*. However, when evaluating the risks posed by sites exceeding these screening values the basis upon which they have been derived should be taken into account when considering the scale/magnitude and hence significance of such exceedances.

⁵ Equation 2.4 in Environment Agency (2009b) sets out the usual approach to considering total systemic exposure with two or three route-specific HCVs. However, using this approach for inorganic arsenic would significantly over estimate the contribution to total risk from inhalation exposure because of the different derivation of the oral and inhalation ID.

Arsine

Arsine is potentially more mobile in soil systems than other forms of inorganic arsenic because it is a gas at ambient temperature and pressure. However, arsine is not included in the derivation of the SGV in Table 2 and exposure to arsine has not been considered separately. The two main reasons for this are:

- a lack of quantitative data on the fate/behaviour of arsine in soil gas and ambient air from which to reasonably estimate exposure;
- the consensus of expert opinion that, although naturally occurring arsine gas may form in soil (primarily through microbial activity), its likely concentration will be very low.

See the box for further information about arsine.

Bioaccessibility and other site-specific factors

Oral HCVs used in the derivation of SGVs are usually based on effects observed in studies where exposure to the chemical of interest was via the diet or drinking water, or by administration by capsule or directly into the stomach. In deriving the SGV there is, therefore, an implicit assumption that the contaminant will be taken up into the body from soil to the same extent as from the medium of exposure in the study used to derive the HCV (Environment Agency, 2005b).

On a site-specific basis, this assumption may not be true. In the case of arsenic, the oral ID is based on epidemiological studies of human exposure to elevated arsenic concentrations in drinking water (Environment Agency, 2009d). Some forms of naturally occurring arsenic may be tightly bound within the soil matrix and pass through the gastro-intestinal system without being released and taken up by the body to the same extent as from water.

Oral bioaccessibility is the fraction of a substance that is released from the soil during processes like digestion into solution, making it available for uptake by the body (Environment Agency and British Geological Survey, 2003a; Environment Agency, 2005a).

Over the past decade, there has been considerable interest from regulators and researchers in developing tests that might enable such reduced biological availability to be robustly measured and taken into account in risk assessment (Ruby *et al.*, 1999; Environment Agency, 2005a, 2005b and 2007b). There has been a particular interest in the UK in arsenic because of the large areas of land where its concentration in soil is naturally elevated (Farago *et al.*, 2003; Nathanail *et al.*, 2004 and 2006; Palumbo-Roe *et al.*, 2005).

The most commonly used site-specific approach to investigate the oral bioaccessibility of arsenic in soil is an *in vitro* test (Environment Agency and British Geological Survey, 2003a, 2003b; Environment Agency,

2006). Two different types of *in vitro* test have been proposed (Environment Agency, 2005a):

- chemical extraction tests that equate to 'easily extractable metals';
- tests that try to mimic biochemical conditions in the human/animal gastrointestinal tract.

Our view is that *in vitro* tests should be used cautiously in assessing risks to health from arsenic in soil since the relationship between measured bioaccessibility and the relative human availability/toxicity of arsenic remains uncertain (Environment Agency, 2005a, 2007b). We are not able to recommend any specific test at this time. However provided such testing has been carried out in

Sources and behaviour of arsine in soil

Arsine (CAS No. 7784-42-1) or arsenic trihydride is a colourless, flammable, non-irritating gas with a mild garlic-like odour (IPCS, 2002). Arsine is produced commercially by the reaction of aluminium arsenide with water or hydrochloric acid, or by electrochemical reduction of other arsenic compounds in acid solutions (IPCS, 2002). It is used extensively in the manufacture of semi-conductors and as a doping agent for silicon-based electronic devices (IPCS, 2002).

There is little information on the environmental behaviour of arsine. Although the toxicity of arsine has been recognised by several expert groups, the general consensus is that environmental exposure to arsine gas will be very low and is likely to be a rare event (EC, 2000; IPCS, 2002; EPAQS, 2008). Arsine is reported to oxidise in ambient air and to decompose rapidly on exposure to light and in contact with moisture (IPCS, 2002; EPAQS, 2008).

Arsine and its methylated compounds may be formed naturally in the environment by microbial transformation of other forms of arsenic including arsenates and arsenites (Woolson, 1977; O'Neil, 1995; Gao and Burau, 1997; IPCS, 2002). Gao and Burau (1997) concluded from earlier studies that evolution of arsine and methylated arsines is much higher from organic arsenicals than from inorganic arsenicals. Woolson (1977) reported that around one per cent of added sodium arsenate was lost from soil in a closed system after 160 days, mainly as dimethylarsine and trimethylarsine.

Gau and Burau (1997) found that only 0.001 and 0.007 per cent of 100 mg kg⁻¹ DW added arsenate and arsenite respectively was lost from a silty clay soil as arsine and methylated arsines over a 70-day period. They concluded that the main controlling factors were the availability of arsenic for biomethylation (including arsenic solubility, soil texture, organic matter content and sorption potential) and its microbial toxicity. A number of other studies have suggested that volatile arsines are produced from lawns and moist soils (O'Neill, 1995). IPCS (2002) cited several studies where the volatilisation rates are reportedly much higher (in the range 12–35 per cent per year) but these studies are much older.

accordance with guidelines for good practice, we consider that the results can be useful as part of a “lines of evidence approach”⁶ to evaluating site-specific risk, including the sensitivity of any quantitative risk assessment.

A list of our publications to date about oral bioaccessibility testing is included in the text box. Other UK and international organisations such as the Bioaccessibility Research Group of Europe (BARGE) have published relevant papers and reports in recent years which may also be useful. This is a rapidly developing field of study and we may revise our view in the future (see our website for any update).

Our publications on oral bioaccessibility testing

- *In-vitro methods for the measurement of the oral bioaccessibility of selected metals and metalloids in soils: a critical review* (Environment Agency and British Geological Survey, 2003a)
- *Measurement of the bioaccessibility of arsenic in UK soils* (Environment Agency and British Geological Survey, 2003b)
- *Report on the international workshop on the potential use of bioaccessibility testing in risk assessment of land contamination* (Environment Agency, 2005b)
- *Questionnaire survey on the use of in-vitro bioaccessibility in human health risk assessment* (Environment Agency, 2006)
- *Inter-laboratory comparison of in-vitro bioaccessibility measurements for arsenic, lead and nickel in soil* (Environment Agency, 2007c)

The availability of arsenic to garden produce depends on a number of complex factors. The soil-to-plant concentration factors are based on a geometric mean value calculated from a review of field observations and experimental studies (Environment Agency, 2009e). In circumstances where the SGV is exceeded and the consumption of produce is indicated as a significant exposure pathway, assessors undertaking a Detailed Quantitative Risk Assessment (DQRA) (Defra and Environment Agency, 2004) may wish to consider the likely forms of arsenic in soil. For example, Farago *et al.* (2003) noted that usually terrestrial plants take up arsenate preferentially over arsenite. Where appropriate, further investigation (including the sampling and chemical analysis of edible parts of fruits and vegetables) could be undertaken to establish site-specific plant concentration factors.

⁶ The lines of evidence approach means that no single piece of evidence such as the outcome of an *in vitro* test should be solely relied on to make a decision about health risks. But alongside other investigations, such as a greater understanding of soil chemistry, *in vitro* tests may inform a site-specific risk evaluation.

Table 3

Contribution to total exposure from soil for the relevant pathways as calculated by the CLEA software

Exposure pathways	Contribution to exposure from soil according to land use (%) ¹		
	Residential	Allotment	Commercial
Ingestion of soil and indoor dust ²	79.9	28.6	94.7
Consumption of homegrown produce and attached soil	7.5	67.1	NA
Dermal contact (indoor)	0.5	NA	1.9
Dermal contact (outdoor)	11.9	4.3	2.8
Inhalation of dust (indoor) ³	0.3	NA	0.6
Inhalation of dust (outdoor) ³	<0.1	0.1	<0.1
Inhalation of vapour (indoor)	NC	NA	NC
Inhalation of vapour (outdoor)	NC	NC	NC
Oral background	NC	NC	NC
Inhalation background	NC	NC	NC

Notes ¹ Rounded to one decimal place.² Treated as one pathway (see Environment Agency, 2009b).³ Included only to illustrate the contribution that this pathway makes to total exposure at the SGV

NA = not applicable (This exposure pathway is not included in the generic land use.)

NC = not calculated (This exposure pathway was not included for chemical-specific reasons.)

Table 4

Recommended chemical data for inorganic arsenic (at 10°C unless stated)

Chemical property	Arsenic	
Air–water partition coefficient, dimensionless	NA	
Dermal absorption fraction, dimensionless	0.03	Environment Agency (2009b)
Diffusion coefficient in air, $\text{m}^2 \text{s}^{-1}$	NA	
Diffusion coefficient in water, $\text{m}^2 \text{s}^{-1}$	NA	
Octanol–water partition coefficient (log), dimensionless	NA	
Organic carbon–water partition coefficient (log), $\text{cm}^3 \text{g}^{-1}$	NA	
Relative molecular mass, g mol^{-1}	NA	
Soil–water partition coefficient, $\text{cm}^3 \text{g}^{-1}$	500	Environment Agency (2009e)
Vapour pressure, Pa	NA	
Water solubility, mg L^{-1}	1,250,000 (25°C)	Environment Agency (2009e)
Soil-to-dust transport factor, g g^{-1}	0.5	Environment Agency (2009b)
Soil-to-plant concentration factor, mg kg^{-1} FW plant per mg kg^{-1} DW soil		
Green vegetable produce	4.3×10^{-4}	Environment Agency (2009e)
Root vegetable produce	4.0×10^{-4}	Environment Agency (2009e)
Tuber vegetable produce	2.3×10^{-4}	Environment Agency (2009e)
Herbaceous fruit produce	3.3×10^{-4}	Environment Agency (2009e)
Shrub fruit produce	2.0×10^{-4}	Environment Agency (2009e)
Tree fruit produce	1.1×10^{-3}	Environment Agency (2009e)

DW= dry weight

FW = fresh weight

NA = not applicable (The CLEA model does not require these values in the derivation of assessment criteria for inorganic chemicals.)

References

Anon, 2003. *Commission relents on arsenic curbs*. *ENDS Report*, 336, 52-53.

ATSDR, 2007. *Toxicological profile for arsenic*. Atlanta, GA: US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Available from: <http://www.atsdr.cdc.gov/toxprofiles/tp2.pdf> [accessed April 2009].

CCME, 2001. *Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health: Arsenic (inorganic)* [originally published in 1997, updated 2001]. Winnipeg: Canadian Council of Ministers of the Environment.

DEFRA and ENVIRONMENT AGENCY, 2004. *Model procedures for the management of land contamination*. Contaminated Land Report 11. Bristol: Environment Agency.

EC, 2000. *Ambient air pollution by As, Cd and Ni compounds. Position paper*. Brussels: European Commission, Working Group on Arsenic, Cadmium and Nickel Compounds. Available from: http://ec.europa.eu/environment/air/pdf/pp_as_cd_ni.pdf [Accessed April 2009].

ENVIRONMENT AGENCY, 2005a. *Environment Agency's science update on the use of bioaccessibility testing in risk assessment of land contamination*. Bristol: Environment Agency. Available from: http://www.environment-agency.gov.uk/static/documents/2-science_update_1284046.pdf [Accessed April 2009].

ENVIRONMENT AGENCY, 2005b. *Report on the international workshop on the potential use of bioaccessibility testing in risk assessment of land contamination*. Science Report SC040054. Bristol: Environment Agency.

ENVIRONMENT AGENCY, 2006. *Questionnaire survey on the use of in-vitro bioaccessibility in human health risk assessment*. Science Report SC040060/SR1. Bristol: Environment Agency. Available from: http://www.environment-agency.gov.uk/static/documents/Research/scho0906bllw_ee_1493326.pdf [Accessed April 2009].

ENVIRONMENT AGENCY, 2007a. *UK Soil and Herbage Pollutant Survey. Report No. 7: Environmental concentrations of heavy metals in UK soil and herbage*. Bristol: Environment Agency.

ENVIRONMENT AGENCY, 2007b. *In-vitro bioaccessibility testing: current science and way forward (Environment Agency Science Update 2)*. Bristol: Environment Agency. Available from: http://www.environment-agency.gov.uk/static/documents/3-science_update_02_1793841.pdf [Accessed April 2009].

ENVIRONMENT AGENCY, 2007c. *Inter-laboratory comparison of in-vitro bioaccessibility measurements for arsenic, lead and nickel in soil*. Science Report SC040060/SR2. Bristol: Environment Agency. Available from: <http://www.environment-agency.gov.uk/static/documents/1-SCHO0307BMLF-e-e.pdf> [Accessed April 2009].

ENVIRONMENT AGENCY, 2009a. *Using Soil Guideline Values*. Science Report SC050021/SGV introduction. Bristol: Environment Agency.

ENVIRONMENT AGENCY, 2009b. *Updated technical background to the CLEA model*. Science Report SC050021/SR3. Bristol: Environment Agency.

ENVIRONMENT AGENCY, 2009c. *Human health toxicological assessment of contaminants in soil*. Science Report SC050021/SR2. Bristol: Environment Agency.

ENVIRONMENT AGENCY, 2009d. *Contaminants in soil: updated collation of toxicological data and intake values for humans. Inorganic arsenic*. Science Report SC050021/SR TOX1. Bristol: Environment Agency.

ENVIRONMENT AGENCY, 2009e. *Supplementary information for the derivation of SGVs for arsenic*. Science Report SC050021/ Technical Review Arsenic. Bristol: Environment Agency.

ENVIRONMENT AGENCY and BRITISH GEOLOGICAL SURVEY, 2003a. *In-vitro methods for the measurement of the oral bioaccessibility of selected metals and metalloids in soils: a critical review*. R&D Technical Report P5-062/TR/01. Bristol: Environment Agency.

ENVIRONMENT AGENCY and BRITISH GEOLOGICAL SURVEY, 2003b. *Measurement of the bioaccessibility of arsenic in UK soils*. R&D Technical Report P5-062/TR02. Bristol: Environment Agency.

EPAQS, 2008. *Consultation on guidelines for metals and metalloids in ambient air for the protection of human health*. London: Department for Environment, Food and Rural Affairs, Expert Panel on Air Quality Standards. Available from: <http://www.defra.gov.uk/corporate/consult/metals-metalloids/consultation.pdf> [Accessed April 2009].

FARAGO, M.E., KAVANAGH, P.J., LEITE, M.J., MOSSOM, J., SAWBRIDGE, G. and THORNTON, I., 2003. Uptake of arsenic by plants in southwest England. *Biogeochemistry of Environmentally Important Trace Elements*, 835, 115-127.

GAO, S. and BURAU, R.G., 1997. Environmental factors affecting rates of arsenic evolution from and mineralisation of arsenicals in soil. *Journal of Environmental Quality*, 26, 753-763.

IPCS, 2001. *Arsenic and arsenic compounds* (2nd edn.). Environmental Health Criteria 224. Geneva: WHO, International Programme on Chemical Safety. Available from: <http://www.inchem.org/documents/ehc/ehc/ehc224.htm> [Accessed April 2009].

IPCS, 2002. *Arsine: human health aspects*. Concise International Chemical Assessment Document 47. Geneva: WHO, International Programme on Chemical Safety. Available from: <http://www.inchem.org/documents/cicads/cicads/cicad47.htm> [Accessed April 2009].

KABATA-PENDIAS, A. and MUKHERJEE, A.B., 2007. *Trace Elements from Soil to Human*. Berlin: Springer-Verlag.

MERWIN, I., PRUYNE, P.T., EBEL, J.G., MANZELL, K.L. and LISK, D.J., 1994. Persistence, phytotoxicity, and management of arsenic, lead and mercury residues in old orchard soils of New York State. *Chemosphere*, 29, 1361-1367.

NATHANAIL, P., McCAFFREY, C., OGDEN, R., FOSTER, N., GILLET, A. and HAYNES, D., 2004. Uptake of arsenic by vegetables for human consumption: a study of Wellingborough allotment plots. *Land Contamination and Reclamation*, 12, 219-238.

NATHANAIL, P., McCAFFREY, C. and HAYNES, D., 2006. Assessing exposure to pedogenic arsenic contamination at a dwelling in Northamptonshire, UK: a case study. *Soil Use and Management*, 21, 508-517.

O'NEILL, P., 1995. *Mercury*. In *Heavy Metals in Soils* (2nd edn.) (ed. B.J. Alloway). London: Blackie Academic & Professional.

PALUMBO-ROE, B., CAVE, M.R., KLINCK, B.A., WRAGG, J., TAYLOR, H., O'DONNELL, K.E. and SHAW, R.A., 2005. Bioaccessibility of arsenic in soils developed over Jurassic ironstones in eastern England. *Environmental Geochemistry and Health*, 27, 121-130.

RUBY, M.V., SCHOOF, R., BRATTIN, W., GOLDADE, M., POST, G., HARNOIS, M., MOSBY, D.E., CASTEEL, S.W., BERTI, W., CARPENTER, M., EDWARDS, D., CRAGIN, D., and CHAPPELL, W., 1999. Advances in evaluating the oral bioavailability of inorganics in soil for use in human health risk assessment. *Environmental Science and Technology*, 33, 3697-3705.

THORNTON, I., 1994. *Sources and pathways of arsenic in south-west England: health implications*. In *Arsenic Exposure and Health*, Chapter 6, 61-70. Northwood, Middlesex: Science and Technology Letters.

USEPA, 2004. *Risk assessment guidance for Superfund. Volume 1: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)*. Final Report EPA/540/R/99/005. Washington, DC: US Environmental Protection Agency.

WOOLSON, E.A., 1977. Generation of alkylarsines from soil. *Weed Science*, 25, 412-416.

XU, J. and THORNTON, I., 1985. Arsenic in garden soils and vegetable crops in Cornwall, England: implications for human health. *Environmental Geochemistry and Health*, 7, 131-133.

Publication Information

This technical note presents information from Science Project SC050021.

Project manager: Jo Jeffries, Science, Head Office

This work was funded by the Environment Agency's Science Department, which provides scientific knowledge, tools and techniques to enable us to protect and manage the environment as effectively as possible.

Published by: Environment Agency, Rio House, Waterside Drive, Aztec West, Almondsbury, Bristol BS32 4UD.

© Environment Agency, May 2009

Author(s): Ian Martin, Róisín De Burca¹ and Hannah Morgan¹

Research Contractor:

¹ Atkins Ltd, The Axis, 10 Holliday Street, Birmingham, B1 1TF, 0121 483 5870

Dissemination Status:

Released to all regions
Publicly available

Legal Status and Disclaimer

The CLEA Guidance incorporates the following

- 1) *Science Report SC050021/SR2: Human health toxicological assessment of contaminants in soil.*
- 2) *Science Report SC050021/SR3: Updated technical background to the CLEA model.*
- 3) *Science Report SC050021/SR4: CLEA Software (Version 1.04) Handbook.*
- 4) *CLEA Software version 1.04 (2009)*
- 5) *Toxicological reports and SGV technical notes*

The CLEA Guidance can help suitably qualified assessors to estimate the risk that a child or adult may be exposed to a soil concentration on a given site over a long period of exposure that may be a cause for concern to human health. The CLEA Guidance does not cover other types of risk to humans, such as fire, suffocation or explosion, or short-term and acute exposures. Nor does it cover risks to the environment or the pollution of water.

The CLEA Guidance is non-statutory. It does not purport to interpret the policies or procedures of the Environment Agency and shall not operate as a statutory

licence, waiver, consent or approval from the Environment Agency. Nothing in the CLEA Guidance shall prejudice, conflict with or affect the exercise by the Environment Agency of its statutory functions, powers, rights, duties, responsibilities, obligations or discretions arising or imposed under the Environment Act 1995 or any other legislative provision enactment, bye-law or regulation.

The CLEA Guidance describes the soil concentrations above which, in the opinion of the Environment Agency, there may be concern that warrants further investigation and risk evaluation for both threshold and non-threshold substances. These levels are a guide to help assessors estimate risk. It does not provide a definitive test for telling when risks are significant.

Regulators are under no obligation to use the CLEA Guidance.